A CASE OF LEFT VENTRICULAR ANEURYSM ASSOCIATED WITH AN ANOMALOUS CORONARY ARTERY

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A 54-year-old man presented with continual angina pectoris at rest, associated with an anomalous coronary artery. He also had an aneurysm at the submitch region of the left ventricular postero-lateral wall, without evidence or prior myocardial infarction, which showed hypokinetic inward motion during systole. We assume that this was a rare case of left ventricular aneurysm without prior myocardial infarction, the etiology which might related to the anomalous coronary artery.

LEFT ventricular aneurysm without prior myocardial infarction is very rare condition. We report here a case of left ventricular aneurysm associated with a non-stenotic anomalous coronary artery in an adult.

CASE REPORT

A 54-year-old man was admitted with a 5-year history of continual angina pectoris at rest, which was relieved easily by sublingual administration of nitroglycerin. His previous health had been good. On examination, he was an intelligent and well-developed man. The pulse rate was 70 beats per minute and regular and his blood pressure was 150/100 mmHg. The jugular venous pulse was normal, and no abnormal impulse was observed on the chest wall. There were no abnormal heart sounds or murmurs.

Chest X-rays revealed an abnormal bulging in the left ventricular contour, which had not changed when compared to an X-ray taken 22 years previously (Fig. 1).

The electrocardiogram at rest showed an axis of -30 degrees, nonspecific intraventricular conduction disturbance and an abnormal Q wave in the aVL lead (Fig. 2). These patterns were similar to those of 8 years ago. During attacks of angina, significant ST depressions and T wave inversions were present in II, III, aVF, V5 and V6 (Fig. 2).

Two-dimensional echocardiography revealed an aneurysm with a narrow orifice at the submitchal region of the left ventricular postero-lateral wall (Fig. 3). The outer wall of the aneurysm was formed by thin myocardium and the pericardium, and showed hypokinetic inward motion during ventricular systole. Relatively thick myocardium which included the antero-lateral papillary muscle formed the inner wall of the aneurysm. There were no mural thrombi in the aneurysm.

Left ventricular angiography obtained by injection of contrast medium into the pulmonary trunk also showed evidence of an aneurysm with a narrow orifice at the postero-lateral wall, and its hypokinetic motion (Fig. 4).

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Fig. 1. Chest X-rays taken when the patient was admitted in 1984 compared with one taken in 1966. Note the similarity in cardiac configuration between two X-rays.

Fig. 2. Electrocardiogram at rest and the taken during an anginal attack.
LV Aneurysm with Anomalous Coronary Artery

Fig. 3. Two-dimensional echocardiograph of the submitial left ventricular aneurysm of the postero-lateral wall. Note the orifice and the double layer structure, consisting of pericardium and outer wall, of the aneurysm. The wall showed inward motion during systole. LV: Left Ventricle, LAO: Left Anterior Oblique, LA: Left Atrium.

Fig. 4. Left ventricular cineangiograms. Contrast medium was injected into the pulmonary trunk. Note that the wall of the posterolateral left ventricular aneurysm showed hypokinetic contraction during systole. ED: End-diastole, ES: End-systole. RAO: Right anterior oblique view, LAO: Left anterior oblique view.

Fig. 5. Radionuclide study of the aneurysm. Note the thallium-201 uptake by the wall. A: Aneurysm.

Fig. 6. Anomalous coronary artery. The left anterior oblique view of the right coronary artery is a compound photograph. See text for explanation. RAO: Right anterior oblique view, LAO: Left anterior oblique view, RCA: Right coronary artery, LCA: Left coronary artery.

A radionuclide study suggested that the myocardium of the wall of the aneurysm was viable because of its positive uptake of thallium-201 (Fig. 5).

These findings indicated that this aneurysm was very different from aneurysms caused by outpouching of the weak portion of the left ventricle (true aneurysm), and also from those caused by myocardial rupture (pseudoaneurysm).

Coronary arteriography revealed an anomalous coronary artery (Fig. 6). The left coronary artery was markedly hypoplastic; consequently the right coronary artery was more prominent, and gave off the posterior descending artery and the bridge
artery running between the aortic root and the pulmonary trunk. The anterior and posterolateral left ventricular wall were supplied by this bridge artery. There was no significant stenosis of the coronary arteries, while the area of the aneurysm was hypovascular. Ergonovine testing was not performed for fear of its effect on blood pressure, which, it was supposed, would speed up the formation of the aneurysm.

DISCUSSION

Previously reported cases of left ventricular aneurysm other than atherosclerotic myocardial infarction are: 1) infections (tuberculosis, syphilis, rheumatic fever, etc.); 2) systemic diseases (sarcoidosis, collagen diseases, etc.); 3) myocarditis or endocarditis; 4) congenital anomaly; 5) anomalous origin of the left coronary artery from the pulmonary artery.

This patient’s clinical profile suggested no infectious, systemic or other definite etiology for the aneurysm. There are three possible explanations for the etiology: 1) sustained myocardial hypoperfusion caused by the anomalous coronary artery and/or repetitive transient myocardial ischemia caused by its vasospastic characteristics; 2) a muscular type of annular submitral-valvular aneurysm\(^2\)\(^-\)\(^4\); and 3) a congenital intramyocardial aneurysm (two chamber left ventricle\(^5\)). We also assume that the intramyocardial dissection mechanism might have played some role in the formation of the aneurysm. Calcium channel blockade was very effective in preventing his anginal attacks, therefore the vasospastic characteristics of the coronary artery, as well as the course of the anomalous bridge artery mentioned above, might have been related to these attacks. However, it is difficult to assign a definite to this aneurysm.

REFERENCES